Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

November 22d, 1897. The President, Dr. Charles W. Burr, in the chair.

ON TABES DORSALIS "ARRESTED BY BLINDNESS."

This was the title of a paper read by Dr. Spiller. He stated that he used the word "arrested" because it had become well known in this connection through the writings of Dejerine, and had been applied to those cases in which the symptoms cease to progress, and even diminish in intensity after blindness appears. He quoted extensively from the statements of Dejerine on this subject, and reported two cases of this form of tabes, in one of which, however, the diagnosis was somewhat doubtful.

One patient had been gradually losing his eyesight during a period of eight years and had complained of pain in the lumbar region and shoulders. His gait was not truly ataxic, and Romberg's sign was not very evident. The patellar reflex in the right leg was much diminished in intensity, and in the left it was scarcely perceptible. Dr. Hansell reported that the patient had tabetic atrophy of both optic nerves. Argyll-Robertson's sign was not present, but the pupils were unequal. The case was interesting on account of the long duration of the gradually developing optic atrophy, without complete blindness even at the present time; the absence of distinct ataxia; the greater involvement of one side of the lumbar cord, as indicated by the difference in the knee-jerks; and the absence of Argyll-Robertson's sign.

The second patient, a negro, gave a clear history of syphilitic infection eight years ago. He had complained of pain in the lumbar region and one thigh, had been losing his vision for two years, and had a girdle sensation and disturbance of micturition. There was no incoördination of gait. The knee-jerks at the first examination were absent, but became visible after the administration of iodide of potassium. Dr. de Schweinitz found reflex iridoplegia, myosis, and divergence of the left eyeball. The optic nerves presented the signs of primary atrophy.

Dr. Spiller spoke of the difficulty of excluding cerebrospinal syphilis in this second case, and of his inability to understand in what way Edinger's substitution theory could be applied to this form of tabes. He thought it would be well to avoid the name of locomotor ataxia as it is very awkward to speak of locomotor ataxia without locomotor ataxia.

Dr. William Osler:—I well remember that Dejerine called special attention to this very point, namely, the absence of ataxic symptoms in those cases affected with early optic atrophy, but I think that it is not quite correct to say that we do not see other instances of preataxic phenomena persisting for a long period, even years, without the development of ataxia; for instance, a localized paralysis of the ocular muscles. I have recently seen an exceptional case in which a diagnosis of locomotor ataxia was made in Paris in 1876. The patient has had lightning-like pains at intervals ever since. He has the Argyll-Robertson pupil and absence of patellar reflex, but he has no ataxia. In this case a diagnosis of locomotor ataxia in the preataxic stage has been made by most of the neurologists of Europe.

Dr. James Hendrie Lloyd:—Such cases are, of course, not unobserved. I have seen one or two, and I think that there is a case somewhat of this type at present in the Philadelphia Hospital. This man has complete optic atrophy, and so far has not the symptoms of posterior sclerosis. Four or five years ago he had several very curious congestive crises, much like what we see in dementia paralytica. Of recent years he has not had even these. He has a distinct history of syphilis.

Brissaud has divided locomotor ataxia into sensory and motor types, and has recorded one or two cases of the sensory type. I think he claims that the fulgurant pains are a common accompaniment of optic atrophy, and also that cases of the sensory type, with optic atrophy and fulgurant pain, without ataxia, are the cases most likely to present spinal arthropathies. This is a point which I am not prepared to confirm from my own observation.

With reference to theories, it may possibly be worth while to recall that the optic nerve-tract is not an ordinary peripheral nerve. It is analogous rather to a tract of the central nervous system. As Monroe has pointed out, it is allied in this way to the posterior columns of the spinal cord. It is probably for this reason that we find it degenerated along with the posterior columns in ordinary cases of tabes. It seems that in some persons the optic nerve-tract suffers first and more than the posterior columns of the cord from the same poison,—syphilis. The reason is obscure, but the fact remains. The only trouble in reaching an explanation is that we do have some spinal symptoms, such as abolition of reflexes and fulgurant pains in these cases of the so-called sensory type.

Dr. Francis X. Dercum:—I think that the title "arrested by blindness" is objectionable. The facts would, perhaps, be better expressed by saying that after optic nerve atrophy appears the symptoms progress very slowly.

The second case which Dr. Spiller reported looks very much like a case of spinal syphilis; especially the return of the knee-jerk after the use of iodide is very suggestive. In the negro, further, locomotor ataxia is excessively rare. I have, however, had one case where the diagnosis was confirmed by autopsy. It is strange that the negro should enjoy such immunity from ataxia, while he does not enjoy a corresponding immunity from paresis.

Dr. Frederick A. Packard showed two brothers, aged ten and eleven years, respectively, out of a family of seven otherwise healthy children, with pseudo-hypertrophic muscular paralysis, changes in the thyroid gland and mental deficiency. The family history was unimportant, save for the existence of goitre, with nervousness and tachycardia—but without exophthalmos—in the mother, and the normal condition of the remaining members of their generation.

Dr. Francis X. Dercum:—Every now and then we see other types of nervous disease in children, which are either accentuated or first make their appearance after an attack of some infectious disease, such as measles. The three cases of cerebral spastic paralysis, shown by me before this Society last winter were an illustration of this fact. This appearance of accentuation after an infection is extremely interesting. It seems as though the power of resistance is occasionally so low in the nervous tissues—perhaps owing to some morphological peculiarity—that under so slight a cause as the toxicity of measles they undergo degeneration.

Dr. William Osler read a paper on paralysis of the hypoglossal nerve.

Dr. Charles K. Mills:—There is one point in Dr. Osler's paper which seems to me to be of special interest, and if I correctly understand the matter, it is doubtful whether his method of reference to accessorius paralysis is correct. In the light of recent observations, it seems to me questionable whether the nerve supply of the palatal and laryngeal muscles can be regarded in any sense as from the spinal accessory nerve. The spinal accessory nucleus has been separated, and probably correctly, from the vagal nucleus and only supplies those muscles regarded as connected with the spinal portion of the nerve. The laryngeal paralysis would not then be referable to the accessorius.

Dr. Francis X. Dercum:—In this connection, I would allude to the morphological fact that in the orang-outang, the vagus supplies the larynx without fusing with the spinal accessory at all.

Dr. Charles W. Burr exhibited

A PORENCEPHALIC BRAIN.

The man from whom the specimen was removed was a negro, thirty-five years of age. Little is known of his previous history, except that he is said to have been an idiot and epileptic all his life. He came to the Philadelphia Hospital some years ago. He suffered from general convulsions and had a spastic gait and spastic rigidity of both arms, and such marked spasm of the tongue and lips that he could not talk. He remained in that condition some years. During the past summer he developed typhoid fever, from which he died.

At the necropsy two large symmetrical cavities were found in the brain, one in each hemisphere. Dr. Burr believed that these were of post-natal origin and regretted that the term porencephaly is used so indiscriminately for cavities in the cerebrum.

Dr. Purves exhibited for Dr. Mills, the brains from two cases of intraventricular hemorrhage. No convulsions had been observed in these cases at the time of the apoplectic seizure.

Dr. Charles K. Mills:—These are two interesting cases, although not of an unusual type. The first was of interest as regards diagnosis in connection with the history of the condition of the kidneys. Dr. Dercum and others in various papers, have called attention to the subject of hemiplegia in nephritis without gross brain lesion. This case presented many features that might have been explained in this way, but my experience has been that when such cases come to autopsy, it is rare not to find a lesion. The cases are also of interest from the absence of convulsions.

The post-mortem notes are worthy of special remark. It is just as important in many of these cases to record the other vascular lesions that are present as it is to record the gross hemorrhage. In many cases of large, sudden, intracerebral hemorrhage lesions are found similar to those which are present in cases of concussion without gross hemorrhage. The symptoms which obscure the diagnosis in some cases are undoubtedly due to the numerous extravasations in various parts of the brain and membranes, and the secondary lesions which result in association with these.

A word with regard to autopsies in brain cases. My attention was called to the method of Dejerine by Dr. Sailer and Dr. Spiller, a year or two ago, and since then I have chiefly followed this method. In many necropsies, the methods adopted render the brain useless for subsequent microscopic and even macroscopic study. An old, and for many purposes, good method is that of Virchow. In this, after opening the ventricles, transections of the brain are made. This is a useful method for rapidly determining the conditions present, and if carefully pursued is a good one, especially for locating lesions in the fresh state.

Another method to which I first called attention is that of entering the brain from below, through the great transverse, calcarine and other fissures and keeping the callosum and parts above intact.

The method of Meynert is well known. In this city a method is sometimes used which seems to be a modification of that of Meynert. After the ganglia are exposed, an incision is carried around them deeply through the brain substance and necessarily through the temporal lobes. Horizontal or vertical sections are then made through the ganglia. This method is open to the objection of destroying the relation of the parts, making it impossible to subsequently examine the brain satisfactorily.

The method of Dejerine, which was described in the postmortem notes of these two cases, is for many purposes a good one. A vertical incision is made, cutting off the occipital lobe. Another vertical incision may or may not be made in front of the basal ganglia. Then an incision is made about on a level with the callosum and the two hemispheres may be separated. In the method of Dejerine the cerebellum, pons and oblongata are removed as a separate specimen by an incision through the pons above the fifth nerve. One advantage of this is that the separate parts can be easily placed together.

A good method of examining the cerebellum is to cut off the lateral lobe on one side, and subsequently the lobe on the other side, keeping the middle lobe as a separate specimen.

Dr. Spiller:—These two cases of intraventricular hemorrhage are further proof of what I recently had occasion to speak of at the Pathological Society, viz., that convulsions are not always observed when hemorrhage into the ventricle occurs, and that they are of doubtful value in diagnosticating such hemorrhage.

Dr. J. P. Arnold, by invitation, exhibited a

TUMOR OF THE CEREBRAL DURA.

The growth had its origin in the dura at the longitudinal fissure. It had displaced the cerebral tissue and interfered with the functions of the leg, arm and face centres.

Dr. James Hendrie Lloyd:—I saw this case in consultation with Dr. Stryker at the Presbyterian Hospital. A diagnosis of brain tumor had been made, but the question was as to the exact location. Looking at the specimen now, it is easy to regret that the man was not operated on. My own belief was that the tumor was in the antero-frontal region. The absence of convulsions, considering the location, is an interesting point. In the absence of distinct localizing symptoms I did not feel justified in recommending operation. There had been no distinct focal or signal epileptoid symptoms at any time. I inquired particularly into this point, and in the absence of such localizing symptoms I did not feel justified in concluding that the growth was in the motor zone. The mere hemiplegia was not enough to indicate this, because hemiplegia is seen in cases of brain tumor in the most widely separated regions. The patient had the peculiar mental inhibition that is sometimes seen in cases of brain tumor, especially in those of the frontal lobes.

Dr. F. X. Dercum:—I understand that the arm and face were more markedly affected than the leg. If so, it is probably to be explained by the way in which the tumor invaded the brain, *i.e.*, from the mesial surface outward, cutting across the fibres for the face and arm passing to the internal capsule. This case reminds me of one which I exhibited before this Society some years ago, in which the tumor had grown downward from the membranes, had separated the frontal from the motor convolutions, and had produced profound symptoms of a general character, but no convulsive seizures.

Dr. Spiller:—The condition of lock spasm which Dr. Arnold has described in this case, as an inability on the part of the patient to relax his hold on an object and a tightening of the grasp when he attempted to relax it, is like a symptom of Thomsen's disease. Schlesinger, of Vienna, told me that he had seen this sign in a case of syringomyelia, and Patrick has recently observed a similar form of muscular contraction in this same disease.